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Health and Environmental Effects Profile for n-Propyl Alcohol

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HEALTH AND ENVIRONMENTAL EFFECTS PROFILE FOR n-PROPYL ALCOHOL

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16. ABSTRACT

The Health and Environmental Effects Profile for n-Propyl Alcohol was prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Solid Waste to support listings of hazardous constituents of a wide range of waste streams under Section 3001 of the Resource Conservation and Recovery Act (RCRA). Both published literature and information obtained from Agency program office files were evaluated as they pertained to potential human health, aquatic life and environmental effects of hazardous waste constituents.

Quantitative estimates have been presented provided sufficient data are available. Existing data are insufficient to determine an Acceptable Daily Intake (ADI) or a carcinogenic potency factor for n-Propyl Alcohol.

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PREFACE

Health and Environmental Effects Profiles (HEEPs) are prepared for the Office of Solid Waste by the Office of Health and Environmental Assessment. The HEEPs are intended to support listings of hazardous constituents of a wide range of waste streams under Section 3001 of the Resource Conservation and Recovery Act (RCRA). Both published literature and information obtained from Agency program office files are evaluated as they pertain to potential human health, aquatic life and environmental effects of hazardous waste constituents.

Quantitative estimates are presented provided sufficient data are available. For systemic toxicants, these include, Acceptable Daily Intakes (ADIs) for chronic exposures. An ADI is defined as the amount of a chemical to which humans can be exposed on a daily basis over an extended period of time (usually a lifetime) without suffering a deleterious effect. In the case of suspected carcinogens, ADIs are not estimated in this document series. Instead, a carcinogenic potency factor or q_1^* is provided. These potency estimates are derived for both oral and inhalation exposures where possible. In addition, unit risk estimates for air and drinking water are presented based on inhalation and oral data, respectively.

The first draft of this document was prepared by Syracuse Research Corporation under EPA Contract No. 68-03-3228. The document was subsequently revised after reviews by staff within the Office of Health and Environmental Assessment: Carcinogen Assessment Group, Reproductive Effects Assessment Group, Exposure Assessment Group, and the Environmental Criteria and Assessment Office in Cincinnati.

The HEEPs will become part of the EPA RCRA docket.

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EXECUTIVE SUMMARY

n-Propyl alcohol is a colorless liquid and is soluble in water. The compound is primarily used as a solvent in different industrial operations. The half-life of n-propyl alcohol vapor in a typical sunlit urban atmosphere has been estimated to be ~45 hours. The most significant fate determining process of n-propyl alcohol in aquatic media is probably its biodegradation. Propanol is produced naturally by plants, microbes, animal waste and volcanos. Although n-propyl alchohol has been detected in some foods, drinking water and gaseous emissions from a whiskey fermentation unit, enough ambient monitoring data is not available to permit an estimation of human exposure to this chemical from inhalation, ingestion or dermal exposure routes.

Propyl alcohol is absorbed through the gastrointestinal tract, lungs and skin. Once absorbed, the parent compound may be excreted or oxidized via an aldehyde intermediate to propionic acid and, subsequently, carbon dioxide and water. It would be expected that a portion of each of the intermediary metabolites would be excreted. Quantitative data on absorption, distribution, elimination and excretion are lacking.

Little information is available on the mammalian toxicology of n-propyl alcohol. However, several subchronic studies indicate that the liver is the primary target organ. Acute exposures result primarily in central nervous system effects. Following exposure to sufficient concentrations, deep narcosis and subsequent death from respiratory failure may result. Some data (abstracts of foreign language papers) exist which indicate that propyl alcohol may be an animal carcinogen. Evaluation of their scientific adequacy was not possible within the framework of available resources.

Acute toxicity testing with fish, larval amphibians, copepods and brine shrimp indicates that 24- to 96-hour LC_{50} concentrations were generally in the range of 2-4 g/ℓ. Exposure to propyl alcohol also inhibited cell multiplication in protozoans (38 mg/ℓ x 72 hours or 568 mg/ℓ x 20 hours), algae (255-3100 mg/ℓ x 8 days) and bacteria (2700 mg/ℓ x 16 hours).

1. INTRODUCTION

n-Propyl alcohol is a clear, colorless, liquid that is made either from ethylene via the oxo process (treatment of ethylene with carbon monoxide to form propanal followed by hydrogenation) or by oxidation of propene (Wocasek, 1968). Propyl alcohol is produced naturally by plants, microbes, animal waste, sewage treatment and volcanos (Graedel, 1978). Some physical parameters are:

CAS Registry Number	71-23-8
Formula	C_3H_8O
Molecular Weight	60.11 (Weast, 1980)
Melting Point	-126.5 (Weast, 1980)
Boiling Point	97.4 (Weast, 1980)
Density	0.8035 at 20°C (Weast, 1980)
Flash Point	96°C (Tag open cup) (Wocasek, 1968)
Vapor Pressure	14.5 torr at 20°C (Wocasek, 1968)
Solubility	Miscible (Weast, 1980)
Production	178,248 pounds in 1980 (USITC, 1981)

Propyl alcohol is used as a solvent in the manufacture of printing inks, nail polishes, polymerization and spinning of acrylonitrile, dyeing of wool, cellulose acetate film, PVC adhesives and metal degreaser; in the manufacture of floor wax and cleaning preparations; as a solvent for resins, cellulose esters, waxes and vegetable oils; and in the manufacturing of brake fluid and antiseptic manufacture (Verschueren, 1977).

2. ENVIRONMENTAL FATE AND TRANSPORT PROCESSES

2.1. AIR

n-Propyl alcohol has been reported to be decomposed in the vapor phase by lower wavelength uv radiation (Wocasek, 1968). Hustert and Parlar (1981) found that 26% of this compound in the gas phase, at a concentration of 100 ppm, decomposed in 2 hours under irradiation at wavelengths >230nm. Although n-propyl alcohol is decomposed by lower wavelength uv radiation (Wocasek, 1968), it is difficult to predict its photochemical reactivity in the troposphere where the available wavelength is >290 nm. The chemical fate of n-propyl alcohol in the atmosphere probably depends on its reactivity with hydroxyl radicals (OH•), oxygen atoms [$O(^3P)$], HO_2 radicals and O_3 molecules. The rate constant for n-propyl alcohol vapor reaction with OH• radicals at 292K is 4.3×10^{-12} cm^3 $molecule^{-1}$ sec^{-1} (Campbell et al., 1976). Assuming the OH• radical concentration of 10^6 radicals cm^{-3}, the half-life of n-propyl alcohol vapor in a typical sunlit urban atmosphere has been estimated to be ~45 hours.

Because n-propyl alcohol is soluble in water, it is likely that this compound, when present in the atmosphere, will be deposited on soils and surface waters via wet precipitation. Some atmospheric n-propyl alcohol may be transferred also to soils and surface waters by way of dry deposition; however, very little information is available in the literature that would facilitate prediction of the quantitative or even the qualitative significance of these transport processes.

2.2. WATER

The most significant fate determining process of n-propyl alcohol in aquatic media is probably its biodegradation. A number of authors have shown that n-propyl alcohol can be biodegraded by isolated microorganisms.

Thus, Pseudomonas sp. (Mehta, 1973), Rhodococcus and Nocardia sp. (Gavrilenko et al., 1979), Fasarium 2P3 (Klausmeier and Jones, 1960), Mycobacterium convolutum (Perry, 1980), a methane using bacterium (Patel et al., 1978), Alcaligenes sp. (Harada and Nagashima, 1975) and M. smegmatic, M. rhodochrous sp. (Lukins and Foster, 1963), all biodegrade n-propyl alcohol.

The biodegradability of n-propyl alcohol with mixed microorganisms was studied by a number of authors. The compound was found to biodegrade with adapted activated sludge (Kalmykova and Rogovskaya, 1978; Pitter, 1976; Rogovskaya et al., 1975), settled domestic sewage (Wagner, 1976; Hatfield, 1957) and activated sludges (Gerhold and Malaney, 1966). The rate of biodegradation of n-propyl alcohol has been determined to be 71 mg/g of activated sludge/hour (Pitter, 1976). Price et al. (1974) have shown that settled domestic sewage can biodegrade n-propyl alcohol when it is present in freshwater and saltwater. In freshwater, 64% of the compound present at an initial concentration of 3-10 mg/ℓ biodegraded in 5 days. In saltwater, only 43% of the compound biodegraded under the same conditions.

Other physiochemical processes, such as evaporation and sedimentation through adsorbed particulate matter, may remove n-propyl alcohol from aquatic media. Very little information is available in the literature that will permit an estimation of the extent of these transport processes for n-propyl alcohol in aquatic media.

If the value of the log octanol/water partition coefficient for n-propyl alcohol is assumed to be the same as that of 2-propanol (0.15) (Konemann, 1981), the log k_{oc}:

$$k_{oc} = \frac{\text{sorption partition coefficient x 100}}{\text{\% organic carbon}}$$

value for this compound can be calculated to be 0.60, based on the correlation equation of Schwarzenbach and Westall (1981). Assuming a sediment

organic content of 0.5% (typical of river sediments) the sediment sorption coefficient would be ~0.02. Thus, the removal of n-propyl alcohol from the aquatic phase by sedimentation may not be a significant transport process.

The rate of evaporation of the compound from the aquatic phase cannot be predicted because n-propyl alcohol is completely miscible in water. However, considering its high solubility, likelihood of forming hydrogen bonds in water, and relatively low volatility, the compound is not expected to evaporate significantly from the aquatic media.

2.3. OTHER MEDIA

The fate and transport of n-propyl alcohol in soil is difficult to predict because of the unavailability of the experimental data. Based on the calculated log k_{oc} value and the solubility of this compound in water, it can be predicted that n-propyl alcohol is likely to leach significantly from soil. No predictions regarding the microbial degradability, volatility and chemical oxidation ability of this compound in soil can be made at this time.

Aliphatic alcohols including n-propyl and isopropyl alcohol were enzymatically converted to their acetate, propionate, n-bulyrate, isovalerate and caproate esters during the incubation with strawberry fruit tissue. The concentration of esters formed seems to depend on the concentration of acids contained in strawberries (Yamashita et al., 1975). Other acid containing fruits are expected to convert n-propyl alcohol to its corresponding esters when the suitable enzyme is available.

3. EXPOSURE

3.1. WATER

The U.S. EPA (1976) reported the presence of n-propyl alcohol in Durham, NC drinking water. The concentration of n-propyl alcohol in the District of Columbia drinking water was estimated to be 1.0 ppb (Scheiman et al., 1974). The lack of more available monitoring data precludes the estimation of exposure to n-propyl alcohol from ingestion of drinking water.

3.2. FOOD

The presence of n-propyl alcohol in Japanese fish lances was reported by Nonaka et al. (1975). n-Propyl alcohol was identified in the fermentation brine of pimentos used for stuffing olives (Nosti and Fernandez, 1975). The estimate of human exposure of n-propyl alcohol through ingestion of foods cannot be made because of the unavailability of more monitoring data.

3.3. INHALATION

Propyl alcohol was detected as one of the air pollutants from grain fermentation units in a whiskey distillery (Carter and Linksy, 1974). Conkle et al. (1975) detected variable concentrations of n-propyl alcohol in the expired air of tobacco smoking individuals. The expired air from non-smoking individuals failed to show any detectable amount of n-propyl alcohol. The origin of n-propyl alcohol in the expired air of smoking individuals was attributed to metabolic origin (Conkle et al., 1975). In the absence of ambient air monitoring data, it is not possible to estimate the human inhalation exposure to n-propyl alcohol.

3.4. DERMAL

Pertinent data regarding dermal exposure to n-propyl alcohol could not be located in the available literature.

4. PHARMACOKINETICS

4.1. ABSORPTION

n-Propyl alcohol is absorbed from the gastrointestinal tract, the lungs and the skin (Browning, 1965). Quantitative data are not available.

4.2. DISTRIBUTION

Once equilibrium is reached, n-propyl alcohol appears to be uniformly distributed with sequestering of the compound or its metabolites not to be expected (Browning, 1965). Quantitative data are not available.

4.3. METABOLISM

Propyl alcohol appears to be metabolized in the same manner as other primary alcohols (Williams, 1959). Oxidation to propionic acid would be expected to proceed via an aldehyde intermediate. The expected metabolite, propionic acid, has been tentatively identified in the blood of treated rabbits. Formation of some lactic acid is also suspected Orskov (1950). By analogy with other primary alcohols, it would be expected that a portion of the propionic acid would undergo further oxidation to carbon dioxide and water (Williams, 1959). Glucuronic acid conjugates have been identified in the urine of treated rabbits and account for <1% of the administered dose (Kamil et al., 1953).

There are two possible pathways of oxidation. Oxidation may proceed either via alcohol dehydrogenase (Lutwak-Mann, 1938; Winer, 1958), or via a microsomal alcohol oxidizing system (Teschke et al., 1974, 1975). This system requires molecular oxygen and NADPH. Catalase - H_2O_2 has a very low affinity for n-propyl alcohol and does not appear to play a role in metabolism (Teschke et al., 1975).

4.4. ELIMINATION

Orskov (1950) studied blood elimination of n-propyl alcohol in rabbits. Animals were treated via intraperitoneal injection. Blood samples were taken 30, 60, 120, 180 and 240 minutes after injection of doses of 0.8, 1.2 or 1.6 g/kg. Blood concentrations are presented in Table 4-1.

The elimination of propyl alcohol appears to follow zero order kinetics as has been shown for ethanol. If the blood concentration data of Orskov (1950) are plotted against time, the blood level time curve is linear with a slope of from 0.0037-0.0058 mg/mℓ/min depending upon which dose data are used.

The elimination of glucuronic acid conjugates has been studied in the rabbit (Kamil et al., 1953). n-Propyl alcohol was given by gavage to three rabbits at a dose of 25 mmol/rabbit. It was determined that 0.9% of the dose was excreted as a conjugate of glucuronic acid. Quantitative determination of other urinary metabolites has not been performed. The data of Orskov (1950) suggest propionic acid as a primary metabolite, although positive identification was not made.

TABLE 4-1

Propyl Alcohol Concentrations (mg %) in Blood*

	Minutes After Injection				
Dose	30	60	120	180	240
0.8 g/kg	70	52	29	14	6
1.2 g/kg	118	89	61	19	9
1.6 g/kg	162	138	121	93	65

*Source: Orskov, 1950

5. EFFECTS

5.1. CARCINOGENICITY

Gibel et al. (1974) tested three analytical grade alcohols including n-propyl alcohol (1-propanol) for carcinogenic and other toxic effects on Wistar rats. The alcohols were redistilled twice and administered via feed tube or subcutaneously to 10-week-old rats of both sexes. The animals were maintained on pellets of a standard feed and had access to water ad libitum. The rats were observed until spontaneous death, at which time they were autopsied and a histological examination of all organs, vertebrate and femur, as well as blood smears and differential leucocyte counts, were conducted. Oral doses of 0.3 mℓ n-propyl alcohol/kg and 1.0 mℓ NaCl/kg solutions were administered 2 times/week to 18 test and 30 control animals, respectively, while 0.06 mℓ n-propyl alcohol or 1.0 NaCl/kg solutions were administered subcutaneously to 31 test and 30 control rats, respectively, also on a 2 times/week basis. Examination of the test animals revealed the presence of a wide spectrum of tumors; however, no breakdown was provided to indicate which tumors were related to treatment with the three aliphatic alcohols tested. The malignant tumors consisted primarily of liver carcinomas and sarcomas, spleen sarcomas, omasum carcinomas, and myelogenous leukemias; although a number of rarer tumors, including a bladder sarcoma, carcinoma of the kidney pelvis, and an adenocarcinoma of the proventriculus were also reported. Benign tumors observed in the test rats were primarily papillomas or papillomatoses of the omasum, with a few reported observations of infiltrative growth, while the benign tumors found in the control animals consisted of small papillomas of the omasum or fibroadenomas of the mammae. A total of 5 malignant and 10 benign tumors were observed in the rats receiving the oral doses of n-propyl alcohol, while 15 malignant and 7

benign tumors were found in the animals receiving the subcutaneous (s.c.) injections. No malignant tumors were found among the controls, and 3 and 2 benign tumors were observed in the control groups administered the oral and s.c. NaCl solutions, respectively. No breakdown of the specific types of tumors were reported for the individual routes of exposure. The authors further noted the observation of a hyperplasia of the hematopoietic parenchyma of the bone marrow, which alternately affected all three hematopoietic cell systems, in addition to an extraosteal metaplasia of the liver and spleen; however, no indication of which of the three alcohols was involved. While it is possible, therefore, to positively attribute both malignant and benign tumors observed in this study to exposure to n-propyl alcohol, it would not be proper to speculate from this paper which types of tumors were related to exposure to 1-propanol. Average survival time of the rats receiving oral treatment was 570 days for the n-propanol treated group, while the respective control rats survived to day 643, at which time they were killed. Subcutaneous administration of 1-propanol resulted in virtually no differences in longevity of survival when compared to control animals. No distinction between sexes was given with respect to either survival time or tumor incidence.

In a continuation study, Gibel et al. (1975) similarly administered doubly distilled n-propyl alcohol twice weekly, either subcutaneously or through use of a probang to Wistar rats of both sexes. Again the animals were 10 weeks old at the start of the experiment and were fed using standard diet cakes and allowed water ad libitum. Blood analyses were conducted for the majority of animals throughout the experiment. The animals were observed until spontaneous death and then subjected to histological examination. As in the 1974 report, controls received 0.9% physiological NaCl

solution, either subcutaneously or by means of a probang. Histological analyses of all organs, vertebrate and femur, as well as blood film preparation and leucocyte-differential blood hemogram and leucocyte count, were conducted. Dosages of n-propyl alcohol used in this experiment were 0.3 mℓ/kg for oral and 0.06 mℓ/kg for s.c. exposures, with average total doses of 50 mℓ and 6 mℓ, respectively, over the course of the study. Controls were again administered dosages of 1.0 mℓ/kg for both treatment routes. These authors again report a wide spectrum of tumors, including some forms such as bladder and kidney pelvis carcinomas, which are noted to be rare in rats. Included in the malignant tumors observed were 2 myelogenous leukemias, 2 liver cell carcinomas, and 2 sarcomas in the oral exposure test group and 1 local sarcoma, 4 myelogenous leukemias, 5 liver sarcomas, 2 spleen sarcomas, 1 renal pelvis carcinoma, 1 bladder carcinoma and 1 uterus carcinoma in the subcutaneously treated group. No malignant tumors were observed in any of the animals from either control group. Administration of n-propyl alochol also resulted in 10 benign tumors and 7 benign tumors in the oral and s.c. test groups, respectively, while only 3 and 2 benign tumors for the the respective oral and s.c. control groups. The benign tumors observed in the test animals were generally papillomas and palilloma-ioses of the anterior stomach section, occasionally with some infiltrative growth, and fibroadenomas of the mammae, although adenomas of the pancreas, the glandular stomach and the adrenal gland, as well as fibromas of the spleen, were observed in at least one animal. However, the authors do not indicate which of the three alcohols (n-propyl, isoamyl or isobutyl) individually tested produced these results. Longevity of the test animals was 570 and 666 days, respectively, for the oral (18 animals) and s.c. (31 animals) exposure groups, as compared with 643 days (time of sacrifice) for both control groups (25 animals each).

The Gibel et al. (1974, 1975) papers as should not necessarily be considered supporting studies, because it was not possible to ascertain whether these two publications represented separate experiments or were merely reports of the same experiment. Because of the remarkably striking similarity (i.e., almost virtually identical) of results and numerically identical tumor incidence reports in the two papers, it appears that only one laboratory experiment was conducted.

Based on the available data, it is difficult to make any definite statement as to the carcinogenic potential of n-propyl alcohol. The results of the Givel et al. papers, however, do suggest that addition research is warrented.

5.2. MUTAGENICITY, TERATOGENICITY AND OTHER REPRODUCTIVE EFFECTS

Pertinent data regarding the mutagenicity, teratogenicity and other reproductive effects of n-propyl alcohol were not located in the available literature.

5.3. CHRONIC TOXICITY

In a long-term study, Gibel et al. (1974) tested three analytical grade alcohols including n-propyl alcohol (1-propanol) for carcinogenic and other toxic effects on Wistar rats. Oral doses of 0.3 mℓ n-propanol/kg were administered 2 times/week to 18 test animals, while 30 control animals received 1.0 mℓ 0.9% NaCl/kg at the same treatment rate. Additionally, a group of 31 test animals was administered s.c. doses of 0.06 mℓ 1-propanol/kg, while 30 control rats received 1.0 mℓ NaCl/kg, both on a 2 times/week schedule. In addition to the neoplastic activity discussed in Section 5.1., changes in the myocardium, such as the appearance of narrow scars resulting from necrosis of the heart muscle, were reported in some animals, as were a few instances of interstitial pancreatitis and fibrosis;

however, the specific aliphatic alcohol(s) producing these effects was/were not noted. Gibel and his colleagues reported that a strong hepatotoxic effect was observed in virtually all animals. Liver steatoses and necroses in varying degrees and some cirrhosis and other undefined hepatic maladies were also observed in the test animals, but the authors do not mention which of the three alcohols produced these responses. Hematotoxic effects, resulting from a hyperplasia of the hematopoietic parenchyma of the bone marrow, were also attributed to n-propyl alochol. Average survival times for the rats are given in Section 5.1.

In a continuation study described in Section 5.1., Gibel et al (1975) similarly administered doubly distilled n-propyl alcohol 2 times/week either subcutaneously or through use of a probang to Wistar rats of both sexes. Dosages of n-propanol used in this experiment were 0.3 mℓ/kg for oral and 0.06 mℓ/kg for s.c. exposures, with average total doses of 50 mℓ and 6 mℓ, respectively, over the course of the study; both controls were administered 1.0 mℓ 0.9% NaCl/kg. In addition to carcinogenic effects discussed in Section 5.1., hepatotoxic and hematotoxic effects were observed. Liver damage ranging from steatosis, stasis and cell necroses to fibrosis and cirrhosis was reported. Hyperplasia of the blood-forming parenchyma of the bone marrow effecting all three hematopoietic cell systems was noted, as was extraosseous metaplasia in the liver and spleen (Gibel et al., 1974). Focal scar formations were observed in the myocardium of some of the test animals, again believed to be due to heart muscle necrosis; interstitial pancreatitis and fibrosis was detected in some instances. However, the specific alcohol(s) producing the heart, pancreas or blood-forming tissue damage was/were not mentioned. Absorption was not discussed and the nontumorigenic results of the oral and s.c. treatment groups were not separated in either

study. The weight of the animals was regularly monitored in the 1975 study, but not mention of any treatment-related gains or losses was made. It was difficult to ascertain whether the Gibel et al. (1974, 1975) papers were merely reports of the same study.

Weese (1928) exposed mice to n-propyl alcohol vapor at concentrations from 0.1-0.2 ccm/15ℓ (mℓ/15ℓ) air. One animal was exposed for 8 days, two for 14 days, one for 24 days. Daily exposure periods varied from 7.5-12 hours. Liver, heart, kidney and lungs were examined histopathologically. The primary effect noted was fatty degeneration of the liver. This occurred in varying degrees in animals exposed for 14 days or longer. These effects were felt to be reversible. A complete translation would be required for thorough evaluation of this study.

In a study to determine whether n-propanol and another congener had greater toxicity than ethanol, Hillbom et al. (1974) provided 1M solutions of the respective alcohols to groups of six 4-month-old Wistar rats as the sole drinking fluid source for 4 months. Control animals received tap water. Ordinary laboratory chow was provided ad libitum for all animals throughout the experiment. Food and fluid consumption, as well as weight gain, were recorded for each animal. All animals were decapitated at the end of the 120-day test period, and liver weights were obtained. The test rats consumed an average of ~5 nmoles n-propanol/100 g bw/day during the treatment period. Histologic examination of the livers of the experimental animals revealed neither an increase in liver fat nor any inflammation, cirrhosis or hepatic steatosis in the livers examined. Throughout the experiment, the amount of n-propanol consumed remained essentially constant and did not significantly influence the food intake of the rats. However, a lower ratio of weight gain to caloric intake in the rats administered

n-propanol was noted (compared to those receiving ethanol). According to the authors, this indicated that the n-propyl alcohol group utilized food less significantly than the ethanol-treated group, but the mechanism by which the n-propyl alcohol interfered with food utilization was not known. The authors pointed out that since the daily doses greatly exceeded those generally consumed in hard liquors and wine by heavy drinkers, but for a much shorter exposure period, no extrapolation to the situation in man could be made. The authors' findings support the view that dietary factors are significant in the pathogenesis of alcohol-induced liver damage (as not dietary imbalances were produced in their experiment and no liver damage was observed). The results of this study are somewhat limited in that although a control group of rats was used; the data obtained from the alcohol-treated groups were apparently never compared with the corresponding parameters of the controls.

5.4. ACUTE TOXICITY

n-Propyl alcohol is relatively low in acute oral toxicity to animals. Inhalation, the more likely route of exposure, may cause irritation of the mucous membranes. n-Propyl alcohol has been shown to be markedly irritating and injurious to the eyes of rabbits, but was neither irritating to the skin nor absorbed in appreciable amounts dermally. Acute exposure to this alcohol may result in mucous membrane irritation, ataxia, lethargy, prostration and narcosis (Rowe and McCollister, 1982).

In acute, lethal exposures to n-propyl alcohol, death is preceded by ataxia, hind limb paralysis, drop in body temperatures, dyspnea and narcosis. Oral exposures also result in gastrointestinal irritation and vomiting (Browning, 1965). A listing of the LD_{50} values for various mammals is given in Table 5-1.

TABLE 5-1

Single-dose Oral Toxicity Values for 1-Propanol in Animals[a]

Species	LD_{50} Values (g/kg)	Reference
Rat	1.87	Smyth et al., 1954
	5.40	IHFA, 1967
	5.35	Golovinskaya, 1976
		Jenner et al., 1964
	6.5	Taylor et al., 1964
Mouse	4.5	Weese, 1928
Rabbit	3.5[b]	Munch and Schwartze, 1925
	2.82	Munch, 1972

[a]Adapted from Rowe and McCollister, 1982

[b]Minimum lethal dose value

Starrek (1938) exposed groups of mice to varying concentrations of n-propyl alcohol in air space. Six groups of mice (two/group) were exposed to n-propyl alcohol levels of 3250 pmm (8 mg/ℓ), 4100 ppm (10 mg/ℓ), 8150 ppm (20 mg/ℓ), 12,250 ppm (30 mg/ℓ), 16,300 ppm (40 mg/ℓ) or 24,500 ppm (60 mg/ℓ) for 480, 240, 135, 120, 90 and 60 minutes, respectively. The time required for the onset of the various symptoms was found to be inversely related to the concentration at which the animals were exposed. Ataxia was observed within 10-14 minutes of exposure to 24,500 ppm, while 90-120 minutes were required for the onset of this symptom at the 3250 ppm exposure level. At the 24,500 ppm concentration, prostration was evident after 19-23 minutes of exposure to n-propyl alcohol vapor, but 165-180 minutes was required for manifestation of prostration at the 3250 ppm concentration. The occurrence of deep narcosis was evident within 60 minutes of exposure to an atmospheric concentration of 24,500 ppm, while 240 minutes was necessary for this effect at 4100 ppm. Death occurred in only 1 of the 12 mice experiencing toxic symptoms. In this same study, another group of mice was exposed to a concentration of 2050 ppm (5 mg/ℓ) and experienced none of the adverse effects observed in the six groups previously described. A separate group of mice survived intermittent exposures to an atmospheric n-propyl alcohol concentration of 7874 ppm (19.3 mg/ℓ) for a total exposure time of 95 hours.

Munch and Schwartze (1925) have determined the minimum oral narcotic dose in rabbits to be 0.0236 mol/kg as opposed to a "certain lethal dose" of 0.0472 mol/kg.

6. AQUATIC TOXICITY

6.1. ACUTE

Acute toxicity tests have been conducted with a brackish water fish (the bleak, Alburnus alburnus L.), freshwater larval amphibians (Mexican axoloti, Ambystoma mexicanum and clawed toad, Xenopus laevis), a brackish water harpacticoid (Nitocra spinipes) and brine shrimp (Artemia salina). The results of these tests, which are summarized in Table 6-1, indicate that LC_{50} concentrations of n-propyl alcohol were generally in the range of 2-4 g/ℓ. Tadpoles have been used to investigate the anesthetic potency of aliphatic alcohols, including n-propyl alcohol. Munch (1972) reported that the threshold narcotic concentration for Rana pipiens tadpoles was 335 mmol/ℓ (~20 g/ℓ); the threshold was determined by the concentration at which touching the tadpoles failed to cause motion. In an earlier study (Overton, 1901) that reportedly used the same criterion, the threshold narcotic concentration was 110 mmol/ℓ (~6.6 g/ℓ). Using the loss of righting reflex in an unspecified tadpole species or the criterion of anesthesia, Pringle et al. (1981) determined an ED_{50} of 5.4×10^{-6} mol/ℓ (~0.32 mg/ℓ). Cell multiplication of the protozoans Entosiphon sulcatum Stein (bacteriovorous flagellate) and Vronema parduczi Chatton-Lwoff (bacteriovorous ciliate) was inhibited by exposure to n-propyl alcohol at concentrations of 38 mg/ℓ (72 hours) and 568 mg/ℓ (20 hours), respectively (Bringmann and Kuehn, 1980a,b).

6.2. CHRONIC

Pertinent data regarding the chronic aquatic toxicity of n-propyl alcohol could not be located in the available literature.

TABLE 6-1

Acute Lethal Effects of Propyl Alcohol to Aquatic Organisms

Species	Duration	Concentration	Method	Effect	Reference
Bleak Alburnus alburnus[a]	96-hour	3-4 g/ℓ	static	LC_{50}	Linden et al., 1979
Creek chub Semotitris a. atromaculatus[b]	24-hour	200-500 ppm	static	critical range[c]	Gillette et al., 1952
Mexican axolotl Ambystema mexicanum[d]	48-hour	4 g/ℓ	static	LC_{50}	Sloof and Baerselman, 1980
Clawed toad Xenopus laevis[d]	48-hour	4 g/ℓ	static	LC_{50}	Sloof and Baerselman, 1980
Harpactacoid Nitocra spinipes[a]	96-hour	2.3 g/ℓ	static	LC_{50}	Linden et al., 1979
Brine shrimp Artemia salina	24-hour	4.2 g/ℓ	static	LC_{50}	Price et al., 1974

[a]brackish water species

[b]freshwater species

[c]"critical range" was defined as the concentration above which four test fish died in 24 hours, and below which all four test fish survived 24 hours.

[d]3- to 4-week old larvae

6.3. PLANT EFFECTS

Bringmann and Kuehn (1978, 1980a) determined that cell multiplication of Microcystis aeruginosa (blue-green alga) and Scenedesmus quadricauda (green alga) was inhibited by exposure to 255 mg/ℓ n-propyl alcohol, respectively, for 8 days.

6.4. RESIDUE

Pertinent experimental data regarding the aquatic residue of n-propyl alcohol could not be located in the available literature. However, Veith et al. (1980) have suggested the use of the following equation to estimate bioconcentration factors (BCFs):

$$\log BCF = 0.76 \log K_{ow} - 0.23$$

where K_{ow} is the octanol/water partition coefficient. Assuming that the log K_{ow} for n-propyl alcohol is similar to the value (0.15) reported for 2-propyl alcohol (Kōneman, 1981), the BCF for whole fish is estimated to be ~0.8. Therefore, significant bioconcentration of this compound is not expected.

6.5. OTHER RELEVANT INFORMATION

Bringmann and Kuehn (1980a) have determined that multiplication of the bacterium Pseudomonas putida was inhibited by exposure to 2700 mg/ℓ n-propyl alcohol for 16 hours.

7. EXISTING GUIDELINES

7.1. HUMAN

A threshold limit value (TLV) of 200 ppm (~500 mg/m^3) has been recommended by the ACGIH (1980). Since no experimental studies are available indicating the concentrations at which a sensory response to n-propyl alcohol vapor is experienced in humans, this TLV has been established by analogy to isopropyl alcohol. At 400 ppm, isopropyl alcohol has been reported to produce mild irritation of the eyes, nose and throat in unaccustomed subjects (Nelson et al., 1943), but would not be expected to produce any detectable narcotic effects at that level (ACGIH, 1980). Since n-propyl alcohol vapor is reportedly considered (ACGIH, 1980) more irritating to the throat than is isopropanol vapor, a TLV between the less toxic isopropyl alcohol and more toxic butyl alcohols was recommended. The USSR and Czechoslovakia suggest limits of 4 and 200 ppm, respectively, for n-propyl alcohol (ACGIH, 1980).

n-Propyl alcohol is also regulated by the U.S. Department of Labor, Occupational Safety and Health Administration (OSHA), 29 CFR 1910.1000. An employee's exposure to n-propyl alcohol shall be limited to 200 ppm or 50 mg/m^3 8-hour time-weighted average (TWA) under this regulation. Other recommendations for good industrial hygiene and medical surveillance practices for n-propyl alcohol are contained in the guidelines published by NIOSH and OSHA.

OSHA believes that all employees that have the potential of exposure in their occupations to hazardous levels of n-propyl alcohol should be screened medically for history of certain conditions which might place the employee at increased risk. OSHA believes that since n-propyl alcohol is a defatting agent and can cause dermatitis on prolonged exposure, that persons with

pre-existing skin disorders may be more susceptible to the effects of this substance. Also, although n-propyl alcohol is not known as a liver toxin in humans, the importance of this organ should be considered before exposing persons with impaired liver function. The same is true for persons having impaired renal function and/or impaired pulmonary function. Since n-propyl alcohol has irritant properties, persons with obstructive airway diseases certainly should not be exposed; breathing the n-propyl alcohol might cause exacerbation of symptoms (Ryer, 1983).

7.2. AQUATIC

Guidelines or standards for the protection of aquatic life were not found in the available literature.

8. APPROACHES TO RISK ASSESSMENT

8.1. INHALATION

There are no data which would be adequate to derive a criterion for inhalation exposures. The TLV has been established to protect against irritation of the throat and therefore would be an inadequate basis for criterion derivations (ACGIH, 1980).

8.2. ORAL

The study of Hillbom et al. (1974) appears to define a free-standing NOAEL. The study was of adequate duration (4 months); however, liver pathology was the only endpoint evaluated. Although the few data available indicate that the liver is expected to be the primary target organ, the potential for other adverse effects has not been investigated adequately.

The reports of Gibel et al. (1974, 1975) indicate a spectrum of adverse effects to several alcohols but do not clearly state which effects are associated with n-propyl alcohol. A thorough review of these studies also indicates that n-propyl alcohol maybe carcinogenic. However, additional data on carcinogenic or other effects of n-propyl alcohol must be obtained before a criterion level can be developed for this chemical.

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